



CLINICAL IMAGES

Tubal pregnancy beyond term

P van der Bijl, T P Mabesa

The rate of ectopic pregnancies has increased 6-fold in the USA and 4-fold in the UK over the past 2 decades.¹ Tubal pregnancies account for 98.3% of all ectopic gestations.² Ectopic pregnancy is a major cause of maternal death during the first trimester, accounting for 9–13% of all pregnancy-associated deaths. Ectopic pregnancy development depends on complex interactions between many factors, although identifiable risk factors are often absent. Many women with the condition will have been symptomatic but often misdiagnosed.^{1,3} Natural progression of ectopic pregnancy usually arrests spontaneously with tubal rupture, tubal abortion, spontaneous resolution or the development of chronicity. Surgical intervention by means of a salpingectomy or a salpingostomy accounts for most others.⁴ Advanced ectopic pregnancy is therefore very rare and often associated with diagnostic difficulties.

A 22-year-old patient was admitted to the obstetric ward with a history of a previous largely uneventful pregnancy. A reactive RPR (rapid plasma reagin) test was found at 18 weeks' gestation and she was treated for syphilis. An absence of fetal movement was reported since 39 weeks' gestation, no fetal heartbeat could be identified with cardiotocography and intra-uterine death was diagnosed. As no ultrasound equipment was available at the referring hospital and an ectopic pregnancy was not diagnosed, several attempts at pharmacological induction were futile. An extra-uterine pregnancy was eventually diagnosed at our hospital by means of ultrasound, accounting for the previous failed attempts at induction. Fetal death was confirmed by absent fetal heartbeat. By this time the pregnancy had progressed to a 'gestation' period of 50 weeks. A left-

sided, tubal pregnancy located at the unruptured fimbrial end, which had progressed to term, was found at laparotomy. A morphologically normal, surprisingly well-preserved dead fetus with moderate areas of cutaneous desquamation, features of oligohydramnios (meconium-stained integument and calcaneovalgus), weighing 3 250 g, was extracted (Fig. 1). The placenta appeared to have derived its blood supply from the fallopian tube.



Fig. 1. The fetus after removal.

Discussion

Most extra-uterine pregnancies are diagnosed in the first trimester and treated using surgical, medical, expectant, or combined strategies. Ectopic pregnancies diagnosed in the second trimester are very rare.⁵ We found only 5 advanced, tubal ectopic pregnancies in the literature⁶ but none that evolved to term and beyond. 98.3% of extrauterine pregnancies occur in the fallopian tube, 79.6% in the ampullary section and 12.3% in the isthmus. Implantation in the fimbrial end and interstitial regions accounts for 6.2% and 1.9% of tubal ectopic pregnancies, respectively.⁷ Early diagnosis of tubal pregnancy is often difficult because of absent or inconsistent clinical symptoms.¹ The diagnosis can be missed in 50% of cases using ultrasound in well-trained units.^{6,8} No vaginal bleeding occurs in 60% of cases when the ectopic pregnancy is unruptured. Less than 50% give a history of risk factors for ectopic pregnancy⁹ including a prior history of ectopic pregnancy, previous tubal surgery (including tubal sterilisation), tubal pathology, history

Pieter van der Bijl qualified MB ChB (cum laude) at Stellenbosch University in 2005. During 2006 he performed his internship at Dhlabeng Regional Hospital, Bethlehem, Free State. He is currently a community service medical officer at Kimberley Hospital in the Northern Cape. He has a keen interest in medical research and has authored/co-authored several papers that have been published in peer-reviewed journals.

Tumelo Mabesa holds BSc and MB ChB degrees from the University of KwaZulu-Natal. During 2006, he was a community service medical officer at Dhlabeng Regional Hospital. He is currently a registrar in radiology at the University of KwaZulu-Natal.

Corresponding author: P van der Bijl (pvdbijl@webmail.co.za)

March 2007, Vol. 97, No. 3 SAMJ



of genital tract infections, infertility and multiple sexual partners.⁵

Our patient perhaps fell into a higher-risk category for an ectopic pregnancy because of the presence of a sexually transmitted disease diagnosed at 18 weeks' gestation. Otherwise, she appeared to have been asymptomatic until 39 weeks, with the usual common presenting signs and symptoms of ectopic pregnancy (eg. abdominal pain/tenderness and vaginal bleeding) being absent. Considering the size of the fetus, it is surprising that the fallopian tube did not rupture. Reasons for the initial misdiagnosis of this tubal pregnancy include the lack of ultrasound equipment and laboratory facilities for determining serum β -HCG and progesterone at the primary care hospital where the patient was first seen.

An antenatal ultrasound is an essential diagnostic and prognostic tool in obstetric practice. All pregnant women

should have a minimum of two such examinations – before 22 weeks' gestation and at 32 weeks' gestation. This would identify cases such as the one described much earlier on.

1. Fylstra DL. Tubal pregnancy: a review of current diagnosis and treatment. *Obstet Gynecol Surv* 1998; 53: 320-328.
2. Chow W, Daling JR, Cates W, Greenberg RS. Epidemiology of ectopic pregnancy. *Epidemiol Rev* 1987; 9: 70-94.
3. Dilbaz S, Caliskan E, Dilbaz B, Degirmenci O, Haberal A. Predictors of methotrexate treatment failure in ectopic pregnancy. *J Reprod Med* 2006; 51: 87-93.
4. Tulandi T, Saleh A. Surgical management of ectopic pregnancy. *Clin Obstet Gynecol* 1999; 42: 31-38.
5. Rackow BW, Quiñones JN, Barnhart K. Second-trimester tubal pregnancy: a case report. *J Reprod Med* 2005; 50: 719-722.
6. Koçak I, Dağdemir A, Yılmaz MS, Üstün. Advanced tubal pregnancy at 30 weeks. *J Obstet Gynaecol Res* 2005; 31: 341-343.
7. Pisarska MD, Carson SA. Incidence and risk factors for ectopic pregnancy. *Clin Obstet Gynecol* 1999; 42: 2-8.
8. Lehner R, Kucera E, Jirecek S, Egarter C, Husslein P. Ectopic pregnancy. *Arch Gynecol Obstet* 2000; 263: 87-92.
9. Stovall TG, Kellerman AL, Ling FW, Buster JE. Emergency department diagnosis of ectopic pregnancy. *Ann Emerg Med* 1990; 19: 1098-1103.

CLINICAL IMAGES

Perforated appendicitis – a rare cause of pneumoperitoneum

David Joseph Ekoué Dosseh, Ayikoé Etienne Ayité, Komla Attipou

Because of its rarity, perforation of the appendix is rarely diagnosed as a cause of pneumoperitoneum. A perforated peptic ulcer is the first differential diagnosis of pneumoperitoneum, but in the socio-epidemiological context of Togo, one should also consider typhoid. In Togo, acute generalised peritonitis is commonly seen with acute appendicitis and complicated typhoid fever. Free air in the peritoneal cavity, which indicates perforation of an intra-abdominal hollow viscus, is usually related to typhoid perforation. Pneumoperitoneum may be seen on a plain abdominal radiograph.

Pneumoperitoneum is described in 75% of peptic perforations and nearly half of colonic perforations,¹ but rarely in appendicular perforations.

There are diagnostic difficulties, and increased postoperative complications may be expected.

Drs Dosseh and Attipou are surgeons in the Department of Digestive Surgery, Teaching Hospital of Tokoin, Lomé, Togo. Dr Ayité is Chief of Digestive Surgery in the same department.

Corresponding author: D Dosseh (davido2k@yahoo.fr)

Case study

A 19-year-old man was hospitalised because of severe diffuse abdominal pain that had commenced 5 days previously, accompanied by vomiting and constipation. An unknown indigenous treatment had not relieved the pain. On admission he was acutely ill and afebrile (37°C) and the abdomen was tender, with generalised guarding, particularly in the hypogastrium. A plain abdominal radiograph revealed a subdiaphragmatic crescent of air (Fig. 1), confirming hollow

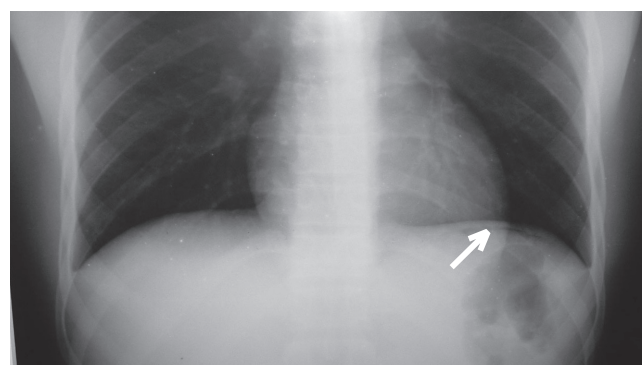


Fig. 1. Plain abdominal radiograph revealing a sub-diaphragmatic crescent of air.



viscus perforation as the cause of peritonitis. The differential diagnosis included typhoid perforation (endemic area, signs pronounced in the hypogastrium with the ileum being the common site for typhoid perforation) and peptic ulcer perforation (self-medication with possible ulcerogenic products and absence of fever indicating chemical peritonitis). Surgery revealed a perforated necrotic zone in the mid-portion of the appendix; there was no visible faecolith and the lumen was not obstructed. Remaining afebrile throughout, the patient made a successful recovery with antibiotic treatment. Histopathological examination revealed inflammation, perforation and a partly necrosed mid-portion of the appendix.

Discussion

Acute appendicitis presents with perforation in about 20 - 30% of cases, and pneumoperitoneum with perforated appendicitis in 0 - 7%.^{2,3} In a review of 6 series, out of 1 192 patients with perforated appendicitis, 14 (1.2%) had pneumoperitoneum.⁴ No pneumoperitoneum was found in 2 000 radiographs in a series of appendicitis.⁴ This rarity of associated pneumoperitoneum is explained by the pathophysiology of acute appendicitis and the anatomical disposition of the appendix.

Luminal air can escape a perforated appendix only if its lumen is patent. One-third of the appendices removed at surgery are completely or partially obstructed.⁴ The mechanism usually responsible for acute appendicitis is obstruction of its lumen by lymphoid hyperplasia secondary to an infection involving the gastrointestinal tract. Less commonly, the obstruction is caused by a faecolith or foreign body.⁵ Luminal obstruction favours bacterial penetration submucosally, transmural suppurative necrosis of the appendicular wall and gangrene and necrosis causing localised or generalised peritonitis. However, because of the luminal obstruction, luminal air does not usually pass into the peritoneal cavity.

Rarely, luminal obstruction is not the primary factor in the pathology of appendicitis. An infection that evolves into suppurative gangrene of the appendicular wall may be haematogenous.⁵ In such cases perforation allows luminal air to escape from the caecum, resulting in a pneumoperitoneum. However, for this leakage to occur freely into the peritoneal cavity, the reactive peritoneal inflammation should not already have walled off the appendix, as frequently occurs efficiently and rapidly.⁴ The appendix should also not be in its retrocaecal position (25% of cases) or subserosal (5% of cases).⁵

Intraperitoneal air may also be caused by gas produced by bacteria in the appendicular abscess,^{3,4} responsible, in our opinion, for the presence of gas within appendicular abscesses rather than in the free peritoneum. Mechanical factors that

favour appendicular perforation have been noted. The frequency of faecoliths, which are usually expelled from the perforation and are usually the cause of appendicular rupture, has been emphasised.⁴ However, a study of 4 950 patients recorded only 126 cases of faecolith (12.2%) among 1 032 cases of perforation.⁶ Obstructive lesions in the caecum may also give rise to appendicular perforation, described in one patient with malignant sigmoid stricture⁴ and in children suffering from severe Hirschsprung's disease.⁷

In our socio-epidemiological context we usually consider typhoid in the differential diagnosis. In the absence of appropriate treatment, surgical complications may occur, most frequently ileal perforation (more than 95% of surgical complications according to Ayité *et al.*⁸). The ileum is the usual site of the typhoid perforation. In a review of the literature on perforated appendicitis associated with peritonitis, the preoperative diagnosis was correct in only 6 of 29 cases.⁴ The clinical features at onset are important, especially right iliac onset of pain.⁴ Radiological signs that point to an appendicular pathology should be sought, namely calcified faecolith, disappearance of the lateral border of the ipsilateral psoas muscle, and replacement of the usual grainy pattern of caecal content with a ground-glass appearance in the right iliac fossa.^{3,4}

However, in spite of the frequency of diagnostic errors, pneumoperitoneum does not seem to alter the prognosis, because its presence expedites surgical intervention.² Pneumoperitoneum has been shown to be an indicator of diffuse peritonitis, constituting a bad prognostic factor,⁴ and there is an increased risk of postoperative complications in cases of perforation.⁶ Possible complications are infectious (parietal abscess, intraperitoneal abscess, enteric fistula, pneumonia, pseudomembranous colitis) or non-infectious (urinary retention, postoperative intestinal obstruction).

Although the association is rare, appendicular perforation must be considered in the differential diagnosis of pneumoperitoneum, principally in the presence of right iliac onset of pain.

1. Le Treut YP. Péritonites. In: Fagniez PL, Houssin D, eds. *Pathologie Chirurgicale: Chirurgie Digestive et Thoracique*. Paris: Masson, 1991: 337-342.
2. Cannova JV, Krummen DM, Nicholson O. Pneumoperitoneum in association with perforated appendicitis. *Am Surg* 1995; **61**: 324-325.
3. Cizmeli MO, Demirag A, Durmus O, Ilgit E. Acute appendicitis associated with pneumoperitoneum. *Br J Clin Pract* 1990; **44**: 646-647.
4. Oberlin P, Valette M, Malafosse M. Pneumopéritoine et péritonite appendiculaire. *Gastroenterol Clin Biol* 1981; **5**: 287-289.
5. Chipponi J. Appendicites. In: Fagniez PL, Houssin D, eds. *Pathologie Chirurgicale: Chirurgie Digestive et Thoracique*. Paris: Masson, 1991: 253-260.
6. Hale DA, Molloy M, Pearl R, Schutt DC, Jaques DP. Appendectomy. A contemporary appraisal. *Ann Surg* 1997; **225**: 252-261.
7. Newman B, Nussbaum A, Kirkpatrick JA jun., Colodny A. Appendiceal perforation, pneumoperitoneum, and Hirschsprung's disease. *J Pediatr Surg* 1988; **23**: 854-856.
8. Ayité A, Etey K, Tchatagba K, *et al.* Cholécystites aiguës althiasiques d'origine typhique. A propos de 5 cas. *La Tunisie Médicale* 1996; **74**: 257-260.